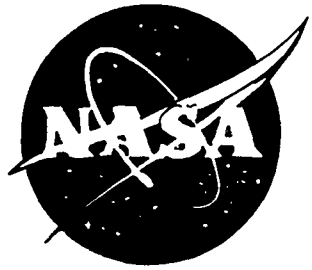
AD 464411

NSAM - 917

SOME RELATIONSHIPS BETWEEN BLOOD ALCOHOL, POSITIONAL ALCOHOL NYSTAGMUS (PAN), AND POSTURAL EQUILIBRIUM (ATAXIA)

Alfred R. Fregly, Martin Bergstedt, and Ashton Graybiel



JOINT REPORT

UNITED STATES NAVAL SCHOOL OF AVIATION MEDICINE

NATIONAL AERONAUTICS AND SPACE ADMINISTRATION

March 1965

Fregly, A. R. 1965
M. Bergstedt, A. Graybiel
SOME RELATIONSHIPS BETWEEN BLOOD ALCOHOL, POSITIONAL ALCOHOL NYSTAGMUS (PAN), AND POSTURAL EQUILIBRIUM (ATAXIA). NSAM - 917. NASA Order No. R-93. Pensacola, Fla.: Naval School of Aviation Medicine, 17 March.

Equilibrium
Nystagmus
Ataxia

Quantitative relationships were explored between blood alcohol levels, positional alcohol nystagmus (PAN), and postural equilibrium performances measured with a new quantitative ataxia test battery and with a series of clinical-type ataxia tests.

Moderate amounts of 80-proof vodka (1 cc per lb. body wt.;55-100 mg% blood alcohol level) produced appreciable decrements in the postural equilibrium functioning of all thirteen vestibular normal subjects evaluated. Maximum decrements occurred at 60-75 minutes following alcohol intake and were fairly well correlated with the peak blood alcohol levels. But more strikingly, the ataxic responses were in very close agreement with the intensity and duration of the PAN I (intoxication period) responses along the time axis. No systematic relationships between the ataxia test performances and PAN phase II responses were found. Ataxic performances recovered during the PAN II period.

Repetition of the experiment two days later with the same subjects under increased stimulation (100-proof vodka in the same dosage) reproduced the findings generally proportional to the increased stimulus.

Fregly, A. R. 1965
M. Bergstedt, A. Graybiel
SOME RELATIONSHIPS BETWEEN BLOOD ALCOHOL, POSITIONAL ALCOHOL NYSTAGMUS (PAN), AND POSTURAL EQUILIBRIUM (ATAXIA). NSAM - 917. NASA Order No. R-93. Pensacola, Fla.: Naval School of Aviation Medicine, 17 March.

Equilibrium
Nystagmus
Ataxia

Quantitative relationships were explored between blood alcohol levels, positional alcohol nystagmus (PAN), and postural equilibrium performances measured with a new quantitative ataxia test battery and with a series of clinical-type ataxia tests.

Moderate amounts of 80-proof vodka (1 cc per lb. body wt.;55-100 mg% blood alcohol level) produced appreciable decrements in the postural equilibrium functioning of all thirteen vestibular normal subjects evaluated. Maximum decrements occurred at 60-75 minutes following alcohol intake and were fairly well correlated with the peak blood alcohol levels. But more strikingly, the ataxic responses were in very close agreement with the intensity and duration of the PAN I (intoxication period) responses along the time axis. No systematic relationships between the ataxia test performances and PAN phase II responses were found. Ataxic performances recovered during the PAN II period.

Repetition of the experiment two days later with the same subjects under increased stimulation (100-proof vodka in the same dosage) reproduced the findings generally proportional to the increased stimulus.

Fregly, A. R. 1965
M. Bergstedt, A. Graybiel
SOME RELATIONSHIPS BETWEEN BLOOD ALCOHOL, POSITIONAL ALCOHOL NYSTAGMUS (PAN), AND POSTURAL EQUILIBRIUM (ATAXIA). NSAM - 917. NASA Order No. R-93. Pensacola, Fla.: Naval School of Aviation Medicine, 17 March.

Equilibrium
Nystagmus
Ataxia

Quantitative relationships were explored between blood alcohol levels, positional alcohol nystagmus (PAN), and postural equilibrium performances measured with a new quantitative ataxia test battery and with a series of clinical-type ataxia tests.

Moderate amounts of 80-proof vodka (1 cc per lb. body wt.;55-100 mg% blood alcohol level) produced appreciable decrements in the postural equilibrium functioning of all thirteen vestibular normal subjects evaluated. Maximum decrements occurred at 60-75 minutes following alcohol intake and were fairly well correlated with the peak blood alcohol levels. But more strikingly, the ataxic responses were in very close agreement with the intensity and duration of the PAN I (intoxication period) responses along the time axis. No systematic relationships between the ataxia test performances and PAN phase II responses were found. Ataxic performances recovered during the PAN II period.

Repetition of the experiment two days later with the same subjects under increased stimulation (100-proof vodka in the same dosage) reproduced the findings generally proportional to the increased stimulus.

Fregly, A. R. 1965
M. Bergstedt, A. Graybiel
SOME RELATIONSHIPS BETWEEN BLOOD ALCOHOL, POSITIONAL ALCOHOL NYSTAGMUS (PAN), AND POSTURAL EQUILIBRIUM (ATAXIA). NSAM - 917. NASA Order No. R-93. Pensacola, Fla.: Naval School of Aviation Medicine, 17 March.

Equilibrium
Nystagmus
Ataxia

Quantitative relationships were explored between blood alcohol levels, positional alcohol nystagmus (PAN), and postural equilibrium performances measured with a new quantitative ataxia test battery and with a series of clinical-type ataxia tests.

Moderate amounts of 80-proof vodka (1 cc per lb. body wt.;55-100 mg% blood alcohol level) produced appreciable decrements in the postural equilibrium functioning of all thirteen vestibular normal subjects evaluated. Maximum decrements occurred at 60-75 minutes following alcohol intake and were fairly well correlated with the peak blood alcohol levels. But more strikingly, the ataxic responses were in very close agreement with the intensity and duration of the PAN I (intoxication period) responses along the time axis. No systematic relationships between the ataxia test performances and PAN phase II responses were found. Ataxic performances recovered during the PAN II period.

Repetition of the experiment two days later with the same subjects under increased stimulation (100-proof vodka in the same dosage) reproduced the findings generally proportional to the increased stimulus.

SOME RELATIONSHIPS BETWEEN BLOOD ALCOHOL, POSITIONAL ALCOHOL NYSTAGMUS (PAN), AND POSTURAL EQUILIBRIUM (ATAXIA)*

Alfred R. Fregly, Martin Bergstedt, and Ashton Graybiel

Bureau of Medicine and Surgery
Project MR005.13-6001
Subtask 1 Report No. 105

NASA Order No. R-93

Released by

Captain H. C. Hunley, MC USN
Commanding Officer

17 March 1965

*This research was conducted under the sponsorship of the Office of Life Science Programs, National Aeronautics and Space Administration.

U. S. NAVAL SCHOOL OF AVIATION MEDICINE
U. S. NAVAL AVIATION MEDICAL CENTER
PENSACOLA, FLORIDA

SUMMARY PAGE

THE PROBLEM

To explore quantitative relationships between blood alcohol levels, positional alcohol nystagmus (PAN), and postural equilibrium performances measured with a new quantitative ataxia test battery and with a series of clinical-type ataxia tests.

FINDINGS

Moderate amounts of 80-proof vodka (1 cc per lb. body wt.; 55-100 mg% blood alcohol level) produced appreciable decrements in the postural equilibrium functioning of all thirteen vestibular normal subjects evaluated. Maximum decrements occurred at 60-75 minutes following alcohol intake and were fairly well correlated with the peak blood alcohol levels. But more strikingly, the ataxic responses were in very close agreement with the intensity and duration of the PAN I (intoxication period) responses along the time axis. No systematic relationships between the ataxia test performances and PAN phase II responses were found; rather, the ataxic performances improved to virtually complete, if not complete, recovery during the PAN II period.

Repetition of the experiment two days later with the same subjects under increased stimulation (100-proof vodka in the same dosage) reproduced the findings generally proportional to the increased stimulus.

ACKNOWLEDGMENTS

The authors gratefully acknowledge the assistance of the following: Mr. James K. Colehour for the blood samples; Dr. Reid H. Leonard for the blood alcohol determinations; Dr. Vernon C. Bragg for the audiometric evaluations; Dr. Earl F. Miller, II, for counterrolling evaluations; Lt. Mike E. McLeod, MC USN, for threshold caloric determinations; Mr. James C. Sansing for technical assistance with the ENG apparatus; Miss Edna Marques and Mrs. Susie Everett for help with nystagmus data reduction; Mr. Theron L. Trimble for obtaining the bulk of the ataxia test battery data and for his assistance with data reduction, and to the student aviators and volunteer enlisted personnel who undertook conscientiously their roles as subjects.

INTRODUCTION

Few quantitative data are available on the correlation between alcohol intoxication and ataxia (1), which is the main concern of this investigation. Reliable measurements of ataxia were ensured with the recent development of a multidimensional quantitative test battery (6). Determinations of positional alcohol nystagmus (PAN) were included partly because of its clearly defined relation to alcohol intoxication (4) and partly because its significance in relation to ataxia has yet to be demonstrated. For release of PAN, at least one functioning labyrinth must be present (3) and, in persons with normal labyrinths, the minimal value of alcohol in the blood must be greater than 20 mgm% (5). Following a single intoxicating dose of alcohol, PAN I appears in about thirty minutes and lasts about three hours, which extends into the hangover phase. Approximately 1-1/2 hours after cessation of PAN I, nystagmus reappears, beating then in the opposite direction (PAN II), and lasting five to seven hours (4).

PROCEDURE

SUBJECTS

Thirteen well-motivated, volunteer subjects participated. Five subjects were Naval or Marine Corps student aviators with an age range of 21-23. The remaining nine subjects, ages 18-24, were enlisted Naval personnel in the capacity of full-time research subjects.

All subjects were in excellent health, and medical evaluation revealed no significant abnormality; all were free of any vestibular disturbance and of a significant history of auricular difficulties. In addition to meeting the requirement of average or better postural equilibrium and ataxia test performances, they revealed normal responses to counterrolling (8), threshold caloric testing (7), and audiometric evaluation. Moreover, no subject revealed spontaneous or positional vestibular nystagmus upon examination prior to the experiment.

APPARATUS

Electronystagmography

Nystagmus was recorded by means of a direct writing two-channel AC-recorder (Sanborn). The time constant was about two seconds. Electrodes were placed near the lateral canthus of each eye and above and below the left eye. A ground electrode was placed on the forehead. All recordings were made with subjects' eyes closed (5).

Test Battery (Short Version)*

Walk H/T (with eyes open, heel-to-toe) rail and Stand E/O (eyes open) rail: metal construction, 8 feet long, 3/4 inches wide, 1-1/2 inches high above the base (3/4" high and 5-1/2" wide), and sand-blasted top surface (Figure 1).

Stand E/C (eyes closed) rail: pinewood construction, 30 inches long, 2-1/4 inches wide, and one inch above its plywood base (also 3/4" high and 5-1/2" wide) on which it was superimposed (Figure 1).

METHOD

Alcohol Stimulus

On an empty stomach, each subject consumed 80-proof vodka in the first experiment and 100-proof vodka in the second experiment (two days later) mixed with orange juice to suit individual taste (about 4:1) in the amount of 1 cc per pound of body weight. To assure a good and rapid absorption, each subject was allowed exactly fifteen minutes to consume the dosage under the following schedule: During the first five minutes subject was permitted to take only small sips; during the next five minutes subject increased his consumption rate steadily; in the final five minutes he consumed steadily the remaining 50 per cent (approximate).

Blood Samples

On each of the experimental days blood was drawn from the cubital vein of each subject several minutes before he consumed alcohol and again at approximately 30 minutes, 60 minutes, 180 minutes, and 270 minutes following the initiation of alcohol intake. Blood alcohol levels were determined utilizing Natelson's microtechniques (9).

Nourishment

Three and one-half hours after start of alcohol intake during each of the experiments, subjects consumed a light lunch. One to two hours preceding lunch, munching on cheese and crackers was permitted.

Experimental Diary

In the interest of obtaining the changing symptomatology along the time axis of the experiment, subjects maintained a printed diary, or log. They were instructed to use the most appropriate terms and qualifiers, such as slight, moderate, severe, and nil, and to note the exact time of appearance and disappearance of symptoms.

- - - - - - - - - -

*A Long Version, which employs six rails of varying widths, from which the Short Version evolved, was designed for similar usage and was described fully with the Short Version in a previous publication (6).

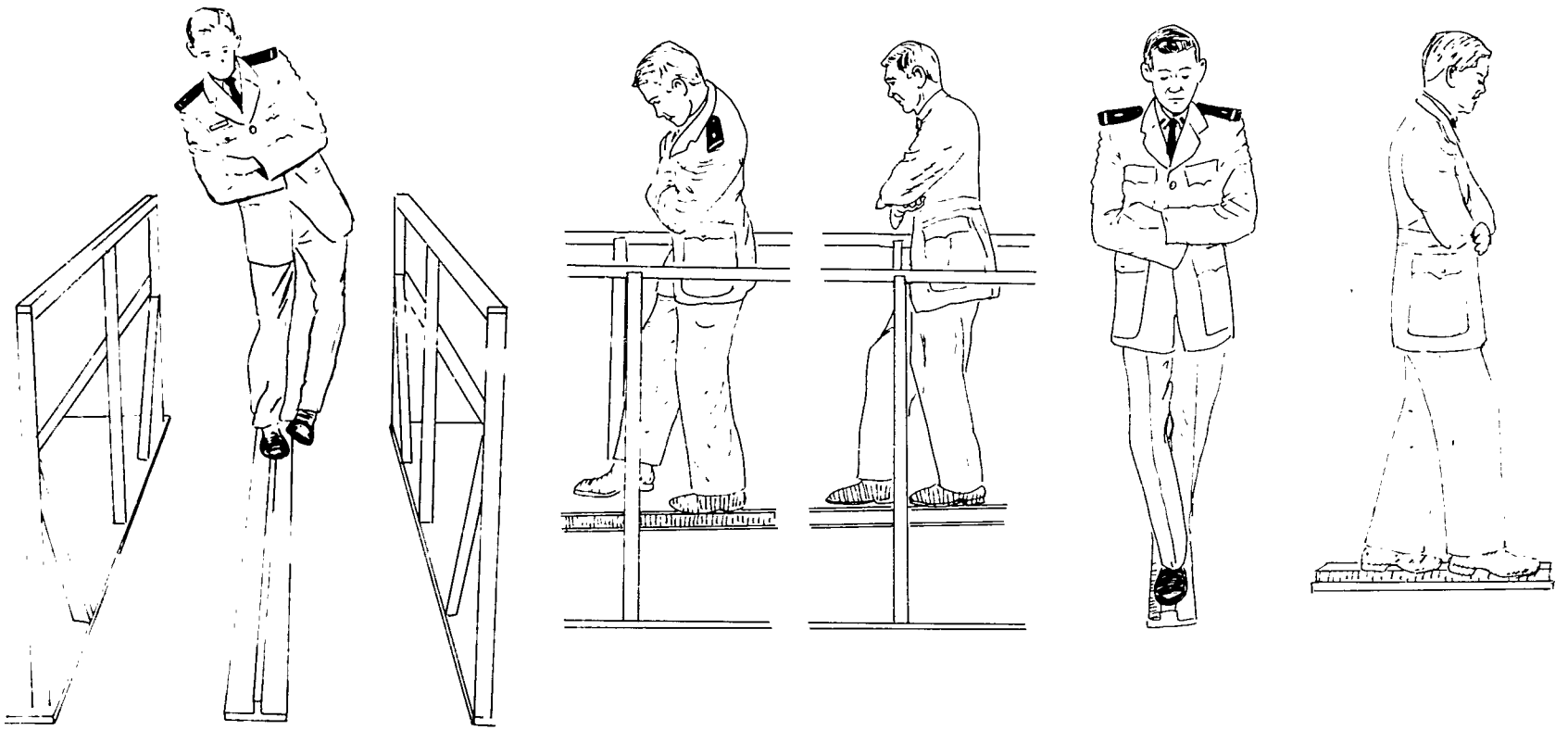


Figure 1

The Test Battery (Short Version): A-B) Walking with eyes open (Walk H/T Test) on the 3/4 inches wide rail; C) Standing with eyes open (Stand E/O Test) on the 3/4 inches wide rail; and D-E) Standing with eyes closed (Stand E/C Test) on the 2-1/4 inches wide rail.

Positional Alcohol Nystagmus

With subjects lying on a couch in a supine position, control (calibration) recordings preceded and followed each of the serial recordings in the left and right lateral head positions. Each lateral position was maintained for at least two minutes and was repeated twice. Then, immediately after disconnection from the recorder, subjects undertook the ataxia tests. In both the 80- and 100-proof experiments*, PAN was recorded in each subject according to the following schedule: 30, 60, 120, 180, 270, 360, 420, and 480 minutes following alcohol intake.

Nystagmus intensity was measured in terms of degrees of eye movement per second. Usually the mean intensity for a ten-second period was measured when PAN reached a peak, i.e.,20-60 seconds after subject's head was turned (5).

Test Battery (Short Version) and Clinical-Type Ataxia Tests#

Shoes were not removed for the test series. Following a combination of written and verbal instructions, all subjects performed all tests in the following sequence:** 1) Sharpened Romberg Test (SR), consisting of standing on the floor with eyes closed for sixty seconds; 2) Test Battery (Short Version) consisting of walking with eyes open (Walk H/T Test) on a 3/4" wide rail; standing with eyes open (Stand E/O Test) on the 3/4" wide rail, and standing with eyes closed (Stand E/C Test) on a 2-1/4" wide rail; 3) standing on one (each) leg for thirty seconds with eyes closed test (SOLEC-R and SOLEC-L); 4) walking a line with eyes closed test (WALEC).

The body position for all tests was as follows: a) body erect or nearly erect, b) arms folded against chest, c) feet in heel-to-toe position and tandemly aligned (SOLEC tests excepted).

The best three out of five trials constituted the scoring of the Test Battery (Short Version), weighted scores were used for the SR and SOLEC tests, and the WALEC was scored in terms of the best two out of three trials (best = least deviant from the line). Maximum scores were as follows: SR: 240 (60 x 4) seconds; Walk H/T: 15 (steps); Stand E/O and Stand E/C tests: 180 (seconds); SOLEC: 150 (30 x 5) seconds; WALEC:## O(inches) deviation.

- - - - - - - - - -

*There were some exceptions, indicated in the results, due to uncontrollable technical difficulties.

#Utmost safety precaution was necessary on the part of the examiner to prevent possible injury of subjects from possible inadvertent falling.

**The administration and scoring procedures utilized have been described in considerable detail previously (6).

##A major limitation of the WALEC procedure is that in notably ataxic individuals the qualitative performance is often more deviant than the individual's score would indicate. Accordingly, the WALEC scores reflected spatial orientation skills more than they reflected ataxia.

Preceding the experiment each subject was retested on each test as often as necessary (usually two to five re-tests) for establishing his peak, or plateau, performance scores. Each subject's best (plateau) performance score on each test (except the WALEC) during pre-experimental testing represents his baseline performance score. The greater variability of the WALEC dictated the use of mean pre-experimental scores of each subject as his baseline performance score.

During the experimental periods subjects undertook these tests at approximately 30, 60, 120, 180, 270, 360, and 420 minutes after the start of alcohol intake.*

RESULTS

BLOOD ALCOHOL LEVELS

A comparison of baseline level with experimental level alcohol concentrations in the blood in the two experimental situations is shown in Figure 2. Maximum mean concentrations of the 80-proof stimulus occurred at about seventy minutes, whereas in the 100-proof experiment maximum mean concentrations occurred earlier, or at sixty minutes following the start of alcohol intake. Moreover, the 80-proof stimulus was sustained in the blood at maximum level only temporarily, whereas the 100-proof was sustained at, or very near, maximum for two hours. Significantly greater concentrations of alcohol in the blood during the 100-proof experiment were evidenced at the 60 minute, 180 minute, and 270 minute periods ($P < .01$, t test).

POSITIONAL ALCOHOL NYSTAGMUS

Before alcohol intake there was no nystagmus in any of the subjects in the three head positions studied, i.e., supine, right lateral, and left lateral.

After alcohol intake all subjects showed positional alcohol nystagmus of varying intensity in right and left lateral positions in accordance with findings reported earlier (4). All subjects also showed the two typical phases of PAN as well as the intermediate period between these two phases.

The mean nystagmus responses are shown in Figure 2A and B, and in terms of the time axis they are similar to the mean responses shown in the remaining figures. Within a given individual there were, however, as has been observed earlier (4), apparent inconsistencies, such as unequal responses and differences in responses both between and within individuals, when the test was repeated. Further variation was found between onset and cessation of a certain phase of nystagmus. Each expected vacillation from zero PAN for a given subject within the PAN I phase was observed. The general pattern of PAN was followed; i.e., PAN I started about one-half hour after alcohol intake and lasted about four hours with nystagmus to the right in the right lateral head position and to the left in the left lateral head position. The supine position produced, usually, either no nystagmus or only single beats.

*Due to uncontrollable technical difficulties, five subjects were not tested during the sixty-minute period of the 80-proof experiment.

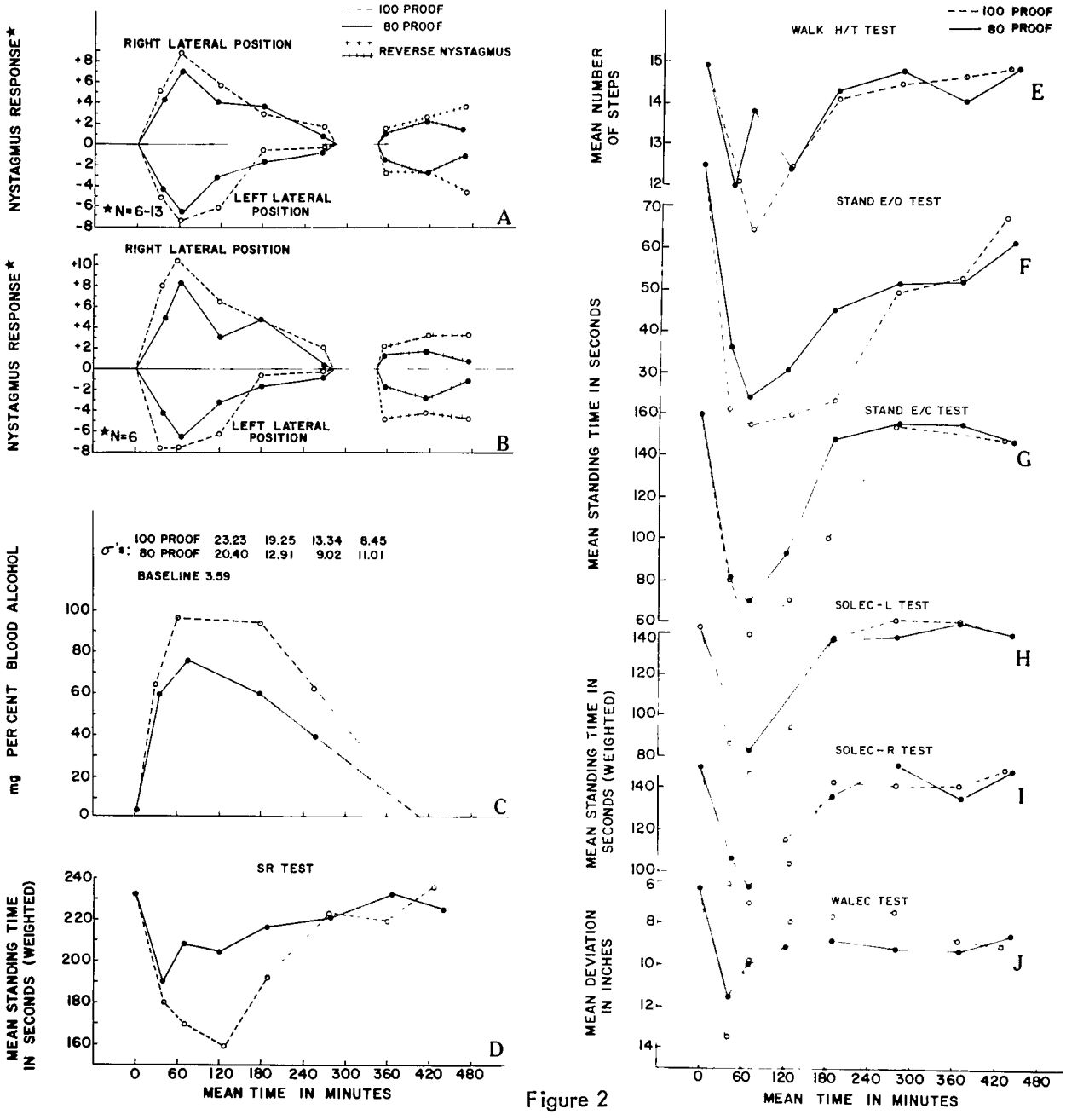


Figure 2

Comparisons of 80-proof with 100-proof alcohol stimulation in thirteen normal male subjects on: A-B) nystagmus intensity; C) blood alcohol level; D) sharpened Romberg test; E) walking with eyes open on a 3/4" wide rail test; F) standing with eyes open on a 3/4" wide rail test; G) standing with eyes closed on a 2-1/4" wide rail test; H-I) standing on one leg with eyes closed-left and right tests; J) walking a line with eyes closed test.

The intermediate period lasted about one hour. PAN II started about five and one-half to six hours after alcohol intake with nystagmus reversed, i.e., to the left in the right lateral position and to the right in the left lateral head position. The recording of phase II was not followed into cessation. The intention was limited to being certain of a clear period of phase II nystagmus responses for comparison on the time axis with the postural equilibrium (ataxia) test findings.

In both the 80- and 100-proof experiments the intensity and variability of PAN phase II was generally less (not statistically significant) than in phase I (Table I). The general tendency was doubly clear and in good agreement with earlier results (4). The goal of establishing the existence of PAN I and II in each subject for comparison with the alcohol-induced ataxia was realized in this investigation.

TEST BATTERY AND CLINICAL-TYPE ATAXIA TESTS

Comparisons of mean baseline performances with mean performances during the two experimental sessions on the Test Battery (Walk H/T, Stand E/O, and Stand E/C) and on the clinical-type ataxia tests are shown in Table II and in Figure 2 D-2J. Statistically significant declines from baseline performance levels were evidenced on all tests as early as 30-45 minutes after alcohol intake. Generally, peak decrements in performance as a result of the alcohol were observed at 60-75 minutes after alcohol intake, and were strikingly in parallel with peak PAN phase I responses and fairly well correlated with peak blood alcohol levels in both experiments. Results in each of the thirteen subjects were remarkably similar to these group results.

Recovery, or near recovery, to baseline levels of performance was observed as early as 120 minutes (WALEC performance) and as late as some 420 minutes (Stand E/O performance) after alcohol intake. Stand E/C performance recovered at between 180-270 minutes ,Walk H/T, SOLEC-R, and SOLEC-L performances recovered at approximately 270 minutes, and SR performance recovered at about 360 minutes after alcohol intake. Generally, the 100-proof vodka produced the greater decrement in performance*, and the slowest recovery to baseline levels of performance. The Test Battery proved to be somewhat more sensitive than the clinical-type tests to alcohol, particularly the Stand E/O Test. Among the clinical-type tests, the SR Test proved to be the most sensitive to the influences of alcohol, although early in each experimental session WALEC Test performances were qualitatively affected considerably more so than the quantitative data indicate. In keeping with expectations, the two dynamic-type or locomotor-influenced tests - Walk H/T and WALEC, were the least influenced by alcohol. Whether or not this finding would hold, however, if a more difficult criterion (e.g., a narrower rail) had been used for the Walk H/T Test and if the qualitatively rich ataxia features of WALEC performance were quantified requires investigation. The progressive recovery of postural equilibrium functioning to virtually complete recovery during the hang-over (PAN phase II) period bore no systematic relationships with PAN phase II responses.

*Only the Stand E/O difference between the 80-proof and 100-proof experiments during Period IV (180 min. after alcohol intake) proved statistically significant ($t = 2.22$, $P.05$).

Table I

Intensity Differences Between 80-Proof and 100-Proof Alcohol-Induced Positional Nystagmus in a Group of 13 Normal Male Subjects

Test Period	Lateral Head Position	Elapsed Time in Minutes 80-Proof Mean	80-Proof S.D.	100-Proof Mean	100-Proof S.D.	Nystagmus Response 80-Proof Mean	80-Proof S.D.	100-Proof Mean	100-Proof S.D.	t of Diff* Between 80-Proof and 100-Proof
I	R					4.3+	2.50	5.0	4.09	1.68
	L	37.8	10.26	33.5	4.55	4.2+	2.82	5.1	4.69	1.64
II	R					7.1#	4.49	8.7	6.22	0.54
	L	64.1	10.09	60.3	2.23	6.6#	3.95	7.4	4.35	0.11
III	R					4.0**	3.91	5.7	4.61	1.07
	L	116.8	7.44	119.2	1.80	3.2**	3.49	6.0	5.81	1.35
IV	R					3.6	3.53	2.9	3.29	0.09
	L	181.5	11.37	180.0	0.00	1.7	2.53	0.6	1.09	0.15
V	R					0.8**	1.40	1.6	1.87	1.57
	L	263.6	19.08	267.8	2.83	0.8**	1.27	0.3	1.29	0.73
VI	R					0.9##	1.60	1.4	2.69	0.43
	L	355.7	3.81	355.5	2.87	1.6	3.34	2.8	3.60	0.52
VII	R					2.1++	1.55	2.5	2.56	0.89
	L	415.0	2.41	417.1	4.75	2.9++	2.13	2.8	4.35	0.60
VIII	R					1.3	2.47	3.5++	2.65	1.40
	L			470.5	9.07	1.2	4.17	4.6++	3.78	1.06

*N = 6; + N = 12; # N = 9; ** N = 11; ++ N = 10; ## Underlined values indicate reverse nystagmus.

Table II

Comparisons of Means and Standard Deviations of Pre-Experimental with Experimental Test Battery and Clinical-Type Ataxia Test Performances and the Significance Levels of Mean Differences in a Group of Thirteen Normal Male Subjects

80-Proof Alcohol (Vodka)

Test Battery and Clinical-Type Ataxia Tests	Baseline		I (42.9)#		II (67.9)+		III (120.7)		IV (187.9)		V (278.8)		VI (367.2)		VII (440.5)	
	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
WALK H/T	14.9	0.85	12.0**	2.45	13.8*	1.09	12.4**	1.60	14.3	1.14	14.8	0.36	14.1	1.49	14.9	0.27
STAND E/O	79.8	40.52	36.1*	43.38	24.3**	22.85	30.4**	37.31	45.1*	33.81	51.6	39.39	52.1	40.24	61.5	51.19
STAND E/C	159.6	38.52	79.9**	46.14	69.0**	49.10	93.3**	56.18	147.5	46.79	154.4	44.99	154.2	46.18	146.2	51.89
SR	232.5	26.11	190.2*	66.25	208.4*	44.20	205.1	64.25	217.1	43.58	221.6	39.33	233.3	16.33	225.8	29.00
SOLEC-R	149.6	1.33	105.8**	39.90	91.1**	47.31	114.4**	35.45	134.7*	28.14	150.0	0.00	134.1*	28.79	147.0	7.17
SOLEC-L	142.5	17.72	102.8**	40.52	82.3**	40.51	105.9**	41.67	135.7	30.76	137.2	23.88	143.2	14.82	136.9	22.12
WALEC	6.3	2.72	11.6*	7.31	10.0	5.81	9.1*	4.57	8.8*	3.98	9.2	4.99	9.3	5.21	8.5	5.62

100-Proof Alcohol (Vodka)

Test Battery and Clinical-Type Ataxia Tests	Baseline		I (41.1)		II (70.4)		III (128.2)		IV (189.2)		V (276.2)		VI (365.8)		VII (427.5)	
	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
WALK H/T	14.9	0.85	12.1**	1.98	10.9**	2.37	12.6*	3.20	14.1*	1.38	14.5	1.08	14.7	0.82	14.9	0.27
STAND E/O	79.8	40.52	20.8**	18.60	17.4**	10.25	19.8**	9.29	33.6**	33.33	49.5*	27.61	53.2	36.00	67.5	50.18
STAND E/C	159.6	38.52	82.1**	39.73	53.2**	36.65	70.9**	43.21	99.8**	57.69	154.3	43.24	150.3	48.61	146.8	51.02
SR	232.5	26.11	180.4*	77.21	170.0*	79.59	159.5**	72.95	192.8	74.91	223.4	40.21	220.2	55.29	236.8	9.57
SOLEC-R	149.6	1.33	93.3**	43.69	83.3**	43.69	102.9**	40.10	142.1	9.52	140.2	20.17	139.8	23.16	147.5	5.89
SOLEC-L	142.5	17.72	86.5**	46.47	71.4**	44.88	93.8**	46.57	136.5	24.82	145.2	8.92	143.5	21.81	138.3	19.00
WALEC	6.3	2.72	13.5**	6.81	9.8	8.04	7.9	4.81	7.6	5.75	7.4	4.60	8.8	5.96	9.0	6.91

*P = .05, **P = .01, (by t test), of mean diff. from baseline mean. #Mean elapsed time from alcohol intake in minutes.
+N=8; Baseline $\bar{x}$'s and σ's were: Walk H/T 14.9,0.33; Stand E/O 90.6,43.11; Stand E/C 172.9,12.53; SR 240.0, 0.00; SOLEC-R 149.4, 1.65; SOLEC-L 144.3, 15.21; WALEC 5.5, 2.83

SUBJECTIVE SYMPTOMATOLOGY

During both experiments all subjects noted in their logs (diaries) that they experienced thirst, hunger, fatigue, drowsiness, and sleepiness. During the 80-proof experiment subjects noted one or more of the following: vertigo, ataxia, muscular incoordination, thick or slurred speech, blurred vision, numbness, headache, lightheadedness, dullness of sensory awareness, cheerfulness, friendliness, lovingness, detachment, depressed feelings, irritability, aggressiveness, nervousness, physical warmth, sweatiness, sour stomach, and recent memory loss. To this list was added the following in the 100-proof experiment: nausea, increased reaction time, feeling of fullness, and slight burning in the stomach. Without exception, all subjects maintained that they experienced somewhat greater to considerably greater intoxication in the 100-proof experiment. However, no subject vomited nor became ill enough to require medical attention. These testimonials by the subjects themselves were consistent with the examiners' observations. Moreover, the severity of the subject-estimated psychophysiological effects were much in accord with the objective blood alcohol levels, PAN responses, and ataxia test responses.

DISCUSSION

Application of the new quantitative ataxia test battery in the present study served the multiple purpose to a degree not possible with any single subjective or objective ataxia test of assessing locomotor versus "static," visual versus nonvisual, and visual-motor versus vestibulo-motor aspects of ataxia in relation to controlled dosages of alcohol. The appreciable performance decrements observed in response to relatively mild stimuli were reliably uniform to a remarkable extent both within and among individuals. Some hierarchical ataxic effects were observed, but inasmuch as the tests were not equated as to difficulty, this finding must be considered tentative at best despite the marked uniformity of these effects in all subjects.

The ataxia test findings related fairly well to the blood alcohol determinations along the time axis of the experiment. Generally, maximum ataxia was observed somewhat sooner (in 40-60 minutes) than the maximum blood alcohol levels (in 60-70 minutes), and, interestingly, the ataxia test performances were on their way to recovery during the periods in which high blood alcohol concentrations were sustained, suggesting that the maximum changes were a dynamic, dependent either on a rising concentration in the tissues or at least the initial rise to a given level.

The striking parallel relationship found between the ataxic test responses and the PAN phase I responses amidst the absence of systematic ataxia test relationships with PAN phase II responses was a major finding, which suggests the workings not only of a common etiological factor but also a common characteristic of this factor.*

*By employing the technique of maintaining the blood alcohol level, while not changing, thereby, the intensity nor the duration of PAN I responses, with repeated administration of small doses of alcohol during the PAN I period (2), the observed ataxia test relationships to PAN I responses in this study might be elaborated or clarified.

But in man, at least, a full understanding of the underlying mechanisms, particularly the relative importance of central versus peripheral factors, governing the relationships between ataxia and the two phases of PAN awaits further investigation. Further studies employing other toxic agents may contribute inestimably to an understanding of the mechanisms or processes underlying ataxia generally and "vestibular ataxia" in particular.

In such studies as the present, which seek normative standards for later comparisons with less than normal performance standards, the importance of utilizing auricular normal subjects in good physical health and free of pathological drinking patterns cannot be over-emphasized for realization of maximum interpretability of results.

REFERENCES

1. Aschan, G., Bergstedt, M., and Dureman, I., Non-titled. In preparation.

2. Aschan, G., Bergstedt, M., and Goldberg, L., Non-titled. In preparation.

3. Aschan, G., Bergstedt, M., and Goldberg, L., Positional alcohol nystagmus in patients with unilateral and bilateral labyrinthine destruction. Confin. Neurol., 24:80-102, 1964.

4. Aschan, G., Bergstedt, M., Goldberg, L., and Luarell, L., Positional nystagmus in man during and after alcohol intoxication. Quart. J. Studies on Alcohol., 17: 381-405, 1956.

5. Bergstedt, M., Studies of positional nystagmus in the human centrifuge. Acta otolaryng., Stockh., Suppl. 165, 1-144, 1961.

6. Graybiel, A., and Fregly, A. R., A new quantitative ataxia test battery. NSAM - 919. NASA Order No. R-93, Pensacola, Fla.: Naval School of Aviation Medicine, 1965.

7. McLeod, M. E. and Meek, J. C., A threshold caloric test: results in normal subjects. NSAM-834. NASA Order No. R-47, Pensacola, Fla.: Naval School of Aviation Medicine, 1962.

8. Miller, E. F., II, and Graybiel, A., A comparison of ocular counterrolling movements between normal persons and deaf subjects with bilateral labyrinthine defects. Ann. Otol., 72:885-893, 1963.

9. Natelson, S., Microtechniques of Clinical Chemistry. Springfield, Ill.: Charles C Thomas, 1957. Pp 198-200.

UNCLASSIFIED
Security Classification

DOCUMENT CONTROL DATA - R&D
(Security classification of title, body of abstract and indexing annotation must be entered when the overall report is classified)

1. ORIGINATING ACTIVITY *(Corporate author)*
Naval School of Aviation Medicine, Pensacola, Florida

2a. REPORT SECURITY CLASSIFICATION
Unclassified

2b GROUP

3 REPORT TITLE
Some Relationships Between Blood Alcohol, Positional Alcohol Nystagmus (PAN), and Postural Equilibrium (Ataxia)

4. DESCRIPTIVE NOTES *(Type of report and inclusive dates)*

5. AUTHOR(S) *(Last name, first name, initial)*
Fregly, A. R., Bergstedt, M., and Graybiel, A.

6. REPORT DATE	7a. TOTAL NO. OF PAGES	7b. NO. OF REFS
17March 1965	12	9

8a. CONTRACT OR GRANT NO.	9a. ORIGINATOR'S REPORT NUMBER(S)
NASA Order No. R-93	NSAM - 917
b. PROJECT NO. MR005.13-6001 Subtask 1	
c.	9b. OTHER REPORT NO(S) *(Any other numbers that may be assigned this report)* 105
d.	

10. AVAILABILITY/LIMITATION NOTICES
Copies of this report may be obtained from Commanding Officer, U. S. Naval School of Aviation Medicine, or from DDC.

11 SUPPLEMENTARY NOTES	12. SPONSORING MILITARY ACTIVITY

13 ABSTRACT

Quantitative relationships were explored between blood alcohol levels, positional alcohol nystagmus (PAN), and postural equilibrium performances measured with a new quantitative ataxia test battery and with a series of clinical-type ataxia tests.

Moderate amounts of 80-proof vodka (1 cc per lb body wt.; 55-100 mg% blood alcohol level) produced appreciable decrements in the postural equilibrium functioning of all thirteen vestibular normal subjects evaluated. Maximum decrements occurred at 60-75 minutes following alcohol intake and were fairly well correlated with the peak blood alcohol levels. But more strikingly, the ataxic responses were in very close agreement with the intensity and duration of the PAN I (intoxication period) responses along the time axis. No systematic relationships between the ataxia test performances and PAN phase II responses were found; rather, the ataxic performances improved to virtually complete, if not complete, recovery during the PAN II period.

Repetition of the experiment two days later with the same subjects under increased stimulation (100-proof vodka in the same dosage) reproduced the findings generally proportional to the increased stimulus.

DD FORM 1 JAN 64 1473

UNCLASSIFIED
Security Classification

14. KEY WORDS	LINK A		LINK B		LINK C	
	ROLE	WT	ROLE	WT	ROLE	WT
Ataxia Postural Equilibrium Tests Positional Alcohol Nystagmus (PAN) Vestibular Functional Tests Blood Alcohol Levels Alcohol-Induced Ataxia						

INSTRUCTIONS

1. ORIGINATING ACTIVITY: Enter the name and address of the contractor, subcontractor, grantee, Department of Defense activity or other organization *(corporate author)* issuing the report.

2a. REPORT SECURITY CLASSIFICATION: Enter the overall security classification of the report. Indicate whether "Restricted Data" is included. Marking is to be in accordance with appropriate security regulations.

2b. GROUP: Automatic downgrading is specified in DoD Directive 5200.10 and Armed Forces Industrial Manual. Enter the group number. Also, when applicable, show that optional markings have been used for Group 3 and Group 4 as authorized.

3. REPORT TITLE: Enter the complete report title in all capital letters. Titles in all cases should be unclassified. If a meaningful title cannot be selected without classification, show title classification in all capitals in parenthesis immediately following the title.

4. DESCRIPTIVE NOTES: If appropriate, enter the type of report, e.g., interim, progress, summary, annual, or final. Give the inclusive dates when a specific reporting period is covered.

5. AUTHOR(S): Enter the name(s) of author(s) as shown on or in the report. Enter last name, first name, middle initial. If military, show rank and branch of service. The name of the principal author is an absolute minimum requirement.

6. REPORT DATE: Enter the date of the report as day, month, year; or month, year. If more than one date appears on the report, use date of publication.

7a. TOTAL NUMBER OF PAGES: The total page count should follow normal pagination procedures, i.e., enter the number of pages containing information.

7b. NUMBER OF REFERENCES: Enter the total number of references cited in the report.

8a. CONTRACT OR GRANT NUMBER: If appropriate, enter the applicable number of the contract or grant under which the report was written.

8b, 8c, & 8d. PROJECT NUMBER: Enter the appropriate military department identification, such as project number, subproject number, system numbers, task number, etc.

9a. ORIGINATOR'S REPORT NUMBER(S): Enter the official report number by which the document will be identified and controlled by the originating activity. This number must be unique to this report.

9b. OTHER REPORT NUMBER(S): If the report has been assigned any other report numbers *(either by the originator or by the sponsor)*, also enter this number(s).

10. AVAILABILITY/LIMITATION NOTICES: Enter any limitations on further dissemination of the report, other than those imposed by security classification, using standard statements such as:

(1) "Qualified requesters may obtain copies of this report from DDC."

(2) "Foreign announcement and dissemination of this report by DDC is not authorized."

(3) "U. S. Government agencies may obtain copies of this report directly from DDC. Other qualified DDC users shall request through _____."

(4) "U. S. military agencies may obtain copies of this report directly from DDC. Other qualified users shall request through _____."

(5) "All distribution of this report is controlled. Qualified DDC users shall request through _____."

If the report has been furnished to the Office of Technical Services, Department of Commerce, for sale to the public, indicate this fact and enter the price, if known.

11. SUPPLEMENTARY NOTES: Use for additional explanatory notes.

12. SPONSORING MILITARY ACTIVITY: Enter the name of the departmental project office or laboratory sponsoring *(paying for)* the research and development. Include address.

13. ABSTRACT: Enter an abstract giving a brief and factual summary of the document indicative of the report, even though it may also appear elsewhere in the body of the technical report. If additional space is required, a continuation sheet shall be attached.

It is highly desirable that the abstract of classified reports be unclassified. Each paragraph of the abstract shall end with an indication of the military security classification of the information in the paragraph, represented as *(TS)*, *(S)*, *(C)*, or *(U)*.

There is no limitation on the length of the abstract. However, the suggested length is from 150 to 225 words.

14. KEY WORDS: Key words are technically meaningful terms or short phrases that characterize a report and may be used as index entries for cataloging the report. Key words must be selected so that no security classification is required. Identifiers, such as equipment model designation, trade name, military project code name, geographic location, may be used as key words but will be followed by an indication of technical context. The assignment of links, roles, and weights is optional.

Fregly, A. R. 1965

M. Bergstedt, A. Graybiel

SOME RELATIONSHIPS BETWEEN BLOOD ALCOHOL, POSITIONAL ALCOHOL NYSTAGMUS (PAN), AND POSTURAL EQUILIBRIUM (ATAXIA). NSAM - 917. NASA Order No. R-93. Pensacola, Fla.: Naval School of Aviation Medicine, 17 March.

Quantitative relationships were explored between blood alcohol levels, positional alcohol nystagmus (PAN), and postural equilibrium performances measured with a new quantitative ataxia test battery and with a series of clinical-type ataxia tests.

Moderate amounts of 80-proof vodka (1 cc per lb. body wt.;55-100 mg% blood alcohol level) produced appreciable decrements in the postural equilibrium functioning of all thirteen vestibular normal subjects evaluated. Maximum decrements occurred at 60-75 minutes following alcohol intake and were fairly well correlated with the peak blood alcohol levels. But more strikingly, the ataxic responses were in very close agreement with the intensity and duration of the PAN I (intoxication period) responses along the time axis. No systematic relationships between the ataxia test performances and PAN phase II responses were found. Ataxic performances recovered during the PAN II period.

Repetition of the experiment two days later with the same subjects under increased stimulation (100-proof vodka in the same dosage) reproduced the findings generally proportional to the increased stimulus.

Equilibrium

Nystagmus

Ataxia

Fregly, A. R. 1965

M. Bergstedt, A. Graybiel

SOME RELATIONSHIPS BETWEEN BLOOD ALCOHOL, POSITIONAL ALCOHOL NYSTAGMUS (PAN), AND POSTURAL EQUILIBRIUM (ATAXIA). NSAM - 917. NASA Order No. R-93. Pensacola, Fla.: Naval School of Aviation Medicine, 17 March.

Quantitative relationships were explored between blood alcohol levels, positional alcohol nystagmus (PAN), and postural equilibrium performances measured with a new quantitative ataxia test battery and with a series of clinical-type ataxia tests.

Moderate amounts of 80-proof vodka (1 cc per lb. body wt.;55-100 mg% blood alcohol level) produced appreciable decrements in the postural equilibrium functioning of all thirteen vestibular normal subjects evaluated. Maximum decrements occurred at 60-75 minutes following alcohol intake and were fairly well correlated with the peak blood alcohol levels. But more strikingly, the ataxic responses were in very close agreement with the intensity and duration of the PAN I (intoxication period) responses along the time axis. No systematic relationships between the ataxia test performances and PAN phase II responses were found. Ataxic performances recovered during the PAN II period.

Repetition of the experiment two days later with the same subjects under increased stimulation (100-proof vodka in the same dosage) reproduced the findings generally proportional to the increased stimulus.

Equilibrium

Nystagmus

Ataxia

Fregly, A. R. 1965

M. Bergstedt, A. Graybiel

SOME RELATIONSHIPS BETWEEN BLOOD ALCOHOL, POSITIONAL ALCOHOL NYSTAGMUS (PAN), AND POSTURAL EQUILIBRIUM (ATAXIA). NSAM - 917. NASA Order No. R-93. Pensacola, Fla.: Naval School of Aviation Medicine, 17 March.

Quantitative relationships were explored between blood alcohol levels, positional alcohol nystagmus (PAN), and postural equilibrium performances measured with a new quantitative ataxia test battery and with a series of clinical-type ataxia tests.

Moderate amounts of 80-proof vodka (1 cc per lb. body wt.;55-100 mg% blood alcohol level) produced appreciable decrements in the postural equilibrium functioning of all thirteen vestibular normal subjects evaluated. Maximum decrements occurred at 60-75 minutes following alcohol intake and were fairly well correlated with the peak blood alcohol levels. But more strikingly, the ataxic responses were in very close agreement with the intensity and duration of the PAN I (intoxication period) responses along the time axis. No systematic relationships between the ataxia test performances and PAN phase II responses were found. Ataxic performances recovered during the PAN II period.

Repetition of the experiment two days later with the same subjects under increased stimulation (100-proof vodka in the same dosage) reproduced the findings generally proportional to the increased stimulus.

Equilibrium

Nystagmus

Ataxia

Fregly, A. R. 1965

M. Bergstedt, A. Graybiel

SOME RELATIONSHIPS BETWEEN BLOOD ALCOHOL, POSITIONAL ALCOHOL NYSTAGMUS (PAN), AND POSTURAL EQUILIBRIUM (ATAXIA). NSAM - 917. NASA Order No. R-93. Pensacola, Fla.: Naval School of Aviation Medicine, 17 March.

Quantitative relationships were explored between blood alcohol levels, positional alcohol nystagmus (PAN), and postural equilibrium performances measured with a new quantitative ataxia test battery and with a series of clinical-type ataxia tests.

Moderate amounts of 80-proof vodka (1 cc per lb. body wt.;55-100 mg% blood alcohol level) produced appreciable decrements in the postural equilibrium functioning of all thirteen vestibular normal subjects evaluated. Maximum decrements occurred at 60-75 minutes following alcohol intake and were fairly well correlated with the peak blood alcohol levels. But more strikingly, the ataxic responses were in very close agreement with the intensity and duration of the PAN I (intoxication period) responses along the time axis. No systematic relationships between the ataxia test performances and PAN phase II responses were found. Ataxic performances recovered during the PAN II period.

Repetition of the experiment two days later with the same subjects under increased stimulation (100-proof vodka in the same dosage) reproduced the findings generally proportional to the increased stimulus.

Equilibrium

Nystagmus

Ataxia